JACC Vol. 9, No. 5
May 1987:989–995

Infarct Artery Perfusion and Changes in Left Ventricular Volume in the Month After Acute Myocardial Infarction

RICHMOND W. JEREMY, MB, ROSEMARY A. HACKWORTHY, MB,
GEORGE BAUTOVICH, MB, PHD, BRIAN F. HUTTON, MSC, PHILLIP J. HARRIS, MB, DPHIL, FACC

Sydney, New South Wales, Australia

The relation between perfusion of the infarct-related artery and changes in left ventricular volume and function during the month after a first myocardial infarction was examined in 40 patients who did not receive thrombolytic therapy. Infarct artery perfusion was documented at predischarge coronary angiography, and left ventricular volume was measured by nongeometric analysis of radionuclide angiograms performed within 48 hours of infarction and at 1 month.

Left ventricular dilation (≥20% increase in volume) developed in 16 patients, whereas 5 patients had a decrease in left ventricular volume of ≥20% by 1 month. Left ventricular dilation occurred in all 14 patients without perfusion of the infarct-related artery, compared with only 2 of 26 patients with perfusion of this artery due to subtotal occlusion or collateral vessels. All five patients whose left ventricular volume decreased by ≥20% had a perfused infarct artery. Multiple linear regression analysis confirmed that the degree of perfusion of the infarct artery (partial r = 0.58, p = 0.001) was a more important predictor of volume change than was infarct size measured by peak creatine kinase (partial r = 0.30, p = 0.009) or QRS score (partial r = 0.20, p = 0.087).

Left ventricular ejection fraction decreased from 0.38 ± 0.10 to 0.30 ± 0.16 (p = 0.05) in 11 patients with an anterior infarct and ventricular dilation; it increased from 0.45 ± 0.10 to 0.62 ± 0.07 (p = 0.02) in the 5 patients with a ≥20% decrease in volume.

The degree of perfusion of the infarct-related artery at predischarge angiography is an important predictor of change in left ventricular volume in the month after infarction, independently of infarct size. Absent perfusion of the infarct artery is associated with the development of left ventricular dilation and impaired left ventricular function.

(J Am Coll Cardiol 1987;9:989–995)

Left ventricular volume is an important prognostic factor in postinfarction patients (1–4). The left ventricular remodeling process which leads to changes in left ventricular volume after myocardial infarction has only recently been studied in detail. This remodeling process begins with expansion of infarcted myocardium (5,6). Left ventricular dilation may be apparent by the time of hospital discharge or may only appear later (7). Dilation of both infarcted and noninfarcted myocardium may continue during the months after infarction (8).

Animal (9–11) and human (5,6,12) studies have demonstrated that infarct expansion and left ventricular dilation are related to infarct size, but the relation is variable, raising the possibility that other factors may be involved (9). The role of chronic coronary stenosis in remodeling of the left ventricle has been questioned (13). A relation between perfusion of the infarct-related artery and the left ventricular remodeling process would have important implications for thrombolytic therapy in acute myocardial infarction.

The aim of this study was to document the relation between perfusion of the infarct-related artery and changes in left ventricular volume and function in the month after a first myocardial infarction in patients who did not receive thrombolytic therapy or coronary angioplasty.

Methods

Study patients. Patients admitted to the coronary care unit with a first myocardial infarction were eligible for the study. Patients with significant valvular heart disease, cardiomyopathy or atrial arrhythmias, and those in whom the first radionuclide angiogram could not be performed within 48 hours of infarction were excluded.

From the Departments of Cardiology and Nuclear Medicine, Royal Prince Alfred Hospital, Sydney, New South Wales, Australia. This study was supported by the National Heart Foundation of Australia, Canberra, Australian Capital Territory, Australia.

Manuscript received June 3, 1986; revised manuscript received November 12, 1986, accepted December 10, 1986.

Address for reprints: Phillip J. Harris, MB, Department of Cardiology, Royal Prince Alfred Hospital, Missenden Road, Camperdown 2050, New South Wales, Australia.

0735-1097/87/$3.50

JACC Vol. 9, No. 5
May 1987:989-995

Forty-five patients were admitted to the study; 5 of them died before 1 month. The remaining 40 patients (37 male and 3 female), with a mean age of 55 years (range 38 to 65), formed the study group. The diagnosis of myocardial infarction was based on a history of chest pain, development of new pathologic Q waves, ischemic T wave changes, or both, on the electrocardiogram and a rise in plasma creatine kinase to at least twice the upper limit of normal (150 units/liter), with an increase in MB fraction on at least one occasion. The study protocol was approved by the Ethics Committee of the hospital and each patient gave informed consent to participate in the study.

Estimation of infarct size. Peak plasma creatine kinase and a QRS score derived from the 12 lead electrocardiogram were used as measures of infarct size. Plasma creatine kinase was measured every 8 hours to the peak level and then daily until it returned to normal levels. If ischemic chest pain recurred or new electrocardiographic changes developed more than 48 hours after infarction, the 8-hourly creatine kinase measurements were repeated to detect possible infarct extension. A standard 12 lead electrocardiogram was recorded daily for the first 4 days and at 10 days and 1 month after infarction.

The electrocardiogram recorded at 10 days was assigned a QRS score based on analysis of Q and R wave patterns according to the method of Palmeri et al. (14). This QRS scoring method is derived from computer simulation of the electrocardiogram and has been validated as a measure of infarct size by anatomic studies (15,16).

Radionuclide studies. All patients underwent equilibrium radionuclide angiography within 48 hours of the onset of infarction and 1 month after infarction. The patient's erythrocytes were labeled by an in vivo method, using an intravenous injection of 10 μg/kg body weight of stannous pyrophosphate (Mallinckrodt Inc.), followed 10 minutes later by 15 to 25 mCi of technetium-99m as pertechnetate (17). The scan image was acquired using a gamma camera with a high sensitivity slant-hole collimator (Sigma 420, Ohio Nuclear). The patient was supine and the collimator aligned in a 30° left anterior oblique position with the slant holes directed caudally. Radionuclide counts were collected for 6 minutes using a matrix size of 64 × 64 pixels and the cardiac cycle was gated into 24 frames using a nuclear medicine computer system (Digital Equipment Corporation Gamma II).

On completion of the scan, a 5 ml sample of peripheral venous blood was collected from the arm that did not receive the technetium-99m injection. This sample was then counted 15 cm above the same collimator for 2 minutes to determine the count rate per milliliter of blood. The volume of blood in the sample was calculated by weighing the syringe before and after blood sampling.

All radionuclide data analysis was performed by experienced nuclear medicine technicians who were unaware of the patient's clinical course or angiographic findings. The end-diastolic and end-systolic regions of interest were automatically defined using an edge detection algorithm (18), with a facility for manual modification by the operator if necessary. A computer-assigned background region, lateral to the end-systolic region and extending around the apex, estimated noncardiac radionuclide activity.

Analysis of left ventricular volume was performed using a count-based nongeometric method (19), which has been shown to correlate closely with measurements of left ventricular volume and cardiac output by contrast angiography and thermodilution studies (20,21). The background-corrected count rate in the left ventricular end-diastolic region was normalized to the count rate per milliliter of the peripheral venous blood, with a correction for radionuclide decay, to yield a radionuclide measure of left ventricular end-diastolic volume.

The formula used for this calculation was:

$$\text{LVEDVrn} = \frac{\text{left ventricular end-diastolic count rate}}{(\text{count rate/ml blood}) \times e^{0.001925t}},$$

where LVEDVrn = radionuclide left ventricular end-diastolic volume; t = time in minutes from midpoint of scan to counting of the peripheral blood sample, and $e^{0.001925}$ = decay rate of technetium-99m activity.

This method yields a radionuclide volume that is smaller than the true left ventricular volume because of attenuation of the technetium-99m photon activity by lung and chest wall tissue intervening between the left ventricular blood pool and the gamma camera. In this study we measured percent change in the attenuated radionuclide volume, rather than attempting conversion of the attenuated volume to true volume by calculating an attenuation factor.

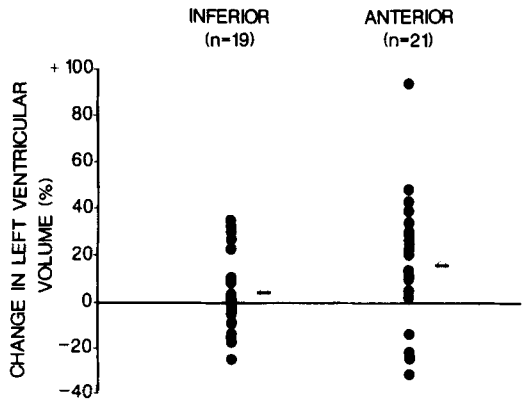
We have previously assessed the reproducibility of this nongeometric method in two patient studies. The reproducibility of the attenuation factor, relating radionuclide to thermodilution measurements of cardiac output and left ventricular stroke volume, was evaluated in serial studies 1 day or 2 weeks apart in 20 subjects in stable condition (22). Individual variations in the relation between radionuclide and thermodilution measurements were $<15\%$ between the two studies. A further study, performed in 12 clinically stable subjects, examined the reproducibility of attenuated radionuclide measurements of left ventricular volume made 1 month apart. There was $<15\%$ variation in left ventricular volume over the month in 11 subjects and 17% variation in the remaining subject. These reproducibility studies indicate that changes in volume of $\geq 20\%$ are unlikely to be due to random variation. Although measured changes in volume of $<20\%$ may also reflect true changes in volume, the possibility of error due to random variation is increased. To avoid overestimation of the occurrence of significant volume changes, only changes $>20\%$ were accepted as real changes in volume.

Coronary angiography. Coronary angiography was performed 7 to 10 days after infarction by either the Judkins or the Sones technique. Each patient had multiple cineangiograms performed in both left and right anterior oblique views with cranial and caudal angulation. All angiograms were reported by at least two experienced angiographers who were unaware of the radionuclide data. The number of vessels with significant stenoses (>70% narrowing of luminal diameter) and the degree of perfusion of the infarct-related artery were recorded. This artery was classified as totally occluded if there was no anterograde filling of the distal segment, or if there was only penetration without perfusion corresponding to grade 0 or 1 in the Thrombolysis in Myocardial Infarction (TIMI) trial (23). The artery was classified as subtotally occluded if there was partial or complete anterograde filling of the distal segment corresponding to grade 2 or 3 perfusion in the TIMI trial. Collateral filling of the infarct artery was also recorded. Good collateral filling was defined according to the guidelines of Hecht et al. (24) as identifiable collateral vessels leading to near normal opacification and rapid washout of the distal segment of the infarct artery. Poor collateral filling was defined as faint opacification and delayed contrast washout from the distal infarct artery.

Clinical data. Each patient's clinical course was reviewed daily during the hospital stay and at 1 month. The occurrence of reinfarction, ventricular arrhythmias or left ventricular failure was noted and the patient's medication recorded.

Data analysis. Changes in left ventricular volume from admission to 1 month were calculated as a percent change relative to the initial volume measurement. A significant change in volume was defined as an increase or decrease of ≥20%. For analysis of the relation between infarct size and change in volume, infarct size was categorized as small, moderate or large according to the peak creatine kinase level and QRS score. Divisions of 1,000 and 2,000 units/liter were used for creatine kinase and 4 and 8 for QRS score

Figure 1. Relation between infarct site and change in left ventricular volume in the month after infarction in 40 patients.



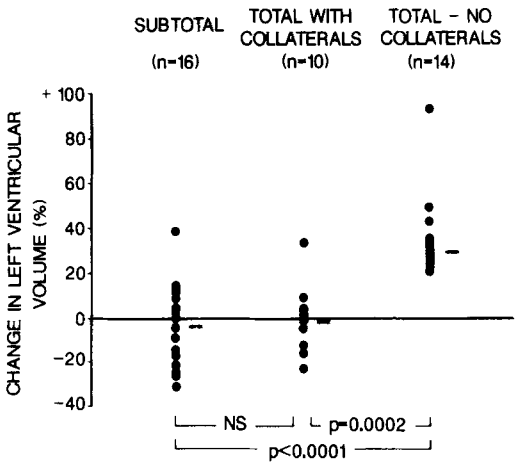


Figure 2. Relation between infarct artery perfusion and change in left ventricular volume in the month after infarction. COLLATERALS = perfusion of a totally occluded artery by collateral vessels; SUBTOTAL = perfusion beyond a subtotal occlusion; TOTAL = total occlusion without collateral filling.

(25). Changes in left ventricular volume were assessed by the Wilcoxon signed-rank test (26). Changes in left ventricular ejection fraction between admission and 1 month were assessed by paired *t* testing. The predictive values of infarct site, infarct size and infarct artery perfusion in relation to changes in left ventricular volume were evaluated by multiple linear regression analysis. Variables with a significant, independent correlation with change in left ventricular volume were identified by stepwise removal of nonsignificant variables, until all remaining variables were significant (27).

Results

Left ventricular volume. During the month after infarction left ventricular volume increased by ≥20% in 16 patients, changed <20% in 19 patients and decreased by ≥20% in 5 patients. Among the 16 patients with ≥20% dilation of the left ventricle were 3 with ≥40% increase in volume. The median change in left ventricular volume for the whole group was an increase of 8% (p = NS).

The relation between infarct site and change in left ventricular volume is illustrated in Figure 1. Left ventricular volume increased by ≥20% in 11 of 21 patients with anterior infarction, compared with 5 of 19 patients with inferior infarction. The median change in volume in the group with anterior infarction was an increase of 18% (p = 0.025), but there was no significant change in the group with inferior infarction.

Infarct artery perfusion. The relation between perfusion of the infarct-related artery and change in left ventricular volume is illustrated in Figure 2. Among 16 patients with perfusion of the infarct artery beyond a subtotal oc-

JACC Vol. 9, No. 5
May 1987:989–995

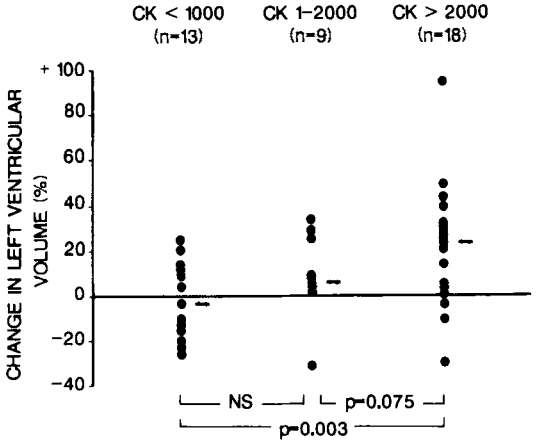
clusion, left ventricular volume increased by ≥20% in one patient and decreased by ≥20% in four patients. There was no significant change for the group. Similarly, among 10 patients with good perfusion of a totally occluded infarct artery through collateral vessels, left ventricular volume increased by ≥20% in only 1 patient and there was no significant change in volume for the group. Thus, left ventricular volume increased in only 2 of 26 patients with perfusion of the infarct artery. In contrast, left ventricular volume increased by 20% or more in all 14 patients with persistent total occlusion of the infarct artery and poor or absent collateral filling, with a median increase in volume of 28% ($p < 0.001$).

The peak creatine kinase level in patients with perfusion of the infarct-related artery through either a subtotal occlusion or good collateral filling was 1,882 ± 1,440 units/liter compared with 2,805 ± 1,545 units/liter in patients with total occlusion without collateral filling, but the difference was not significant. There was also no significant difference in infarct size, measured by QRS score, between patients with and without perfusion of the infarct artery.

Twenty-six patients had one vessel disease and 14 patients had two or three vessel disease. There was no relation between the number of diseased vessels and change in left ventricular volume in the month after infarction.

Infarct size. The relation between infarct size, denoted by peak creatine kinase level, and change in left ventricular volume is illustrated in Figure 3. Left ventricular volume increased by ≥20% in 11 of 18 patients with peak creatine kinase >2,000 units/liter compared with only 5 of 22 patients with peak creatine kinase <2,000 units/liter. The median change in left ventricular volume in patients with peak creatine kinase >2,000 units/liter was an increase of 25% ($p = 0.005$), but there was no significant change among patients with peak creatine kinase <1,000 units/liter or between 1,000 and 2,000 units/liter. Similarly, when patients were grouped according to the QRS score on the electrocardiogram (Fig. 4), the only significant change in volume occurred in patients with a QRS score >7, who had a median increase of 31% ($p = 0.02$).

Figure 3. Relation between infarct size measured by peak creatine kinase (CK) serum level (in units/liter) and change in left ventricular volume in the month after infarction. Significance of differences in volume changes between groups is shown.



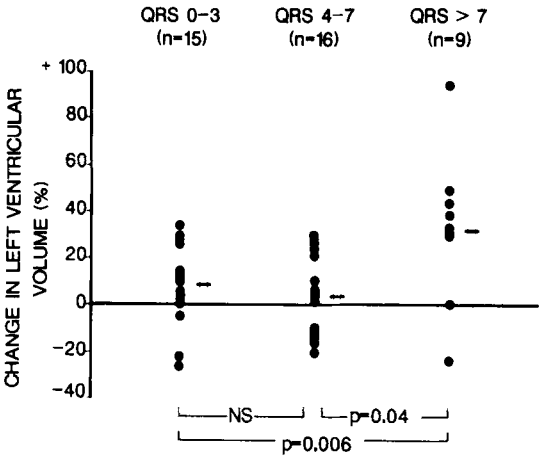


Figure 4. Relation between infarct size measured by QRS score on the 12 lead electrocardiogram and change in left ventricular volume in the month after infarction.

Multivariable analysis. The relation of infarct site, degree of perfusion of the infarct-related artery, peak creatine kinase and QRS score to change in left ventricular volume in the month after infarction was analyzed by multiple linear regression. The overall correlation coefficient with all four variables included in the analysis was 0.75 ($p = 0.0001$). Infarct site and QRS score were not significant and when they were removed from the model the correlation coefficient remained 0.75. Of the two remaining significant, independent predictors of volume change, the degree of infarct artery perfusion had a higher partial correlation coefficient ($r = 0.58$, $p = 0.001$) than did peak creatine kinase (partial $r = 0.30$, $p = 0.009$).

The importance of perfusion of the infarct-related artery as a predictor of change in left ventricular volume, independent of infarct size, was confirmed by substituting QRS score for peak creatine kinase in the two-variable model. The degree of perfusion of the infarct artery remained the variable most closely correlated with change in left ventricular volume, whereas QRS score was marginally significant (partial $r = 0.20$, $p = 0.087$).

The independent relation of perfusion of the infarct-related artery and infarct size as predictors of change in left ventricular volume is illustrated in Figure 5. At each level of infarct size, denoted by peak creatine kinase, there was a greater increase in left ventricular volume in patients without perfusion of the infarct-related artery than in patients with perfusion of this artery through either a subtotal lesion or collateral filling.

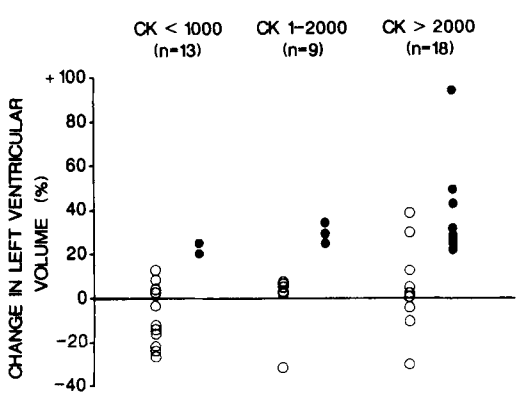
Left ventricular ejection fraction. The relation between changes in left ventricular volume in the month after

infarction and left ventricular ejection fraction is illustrated in Figure 6. Left ventricular ejection fraction increased from 0.45 ± 0.10 to 0.62 ± 0.07 ($p = 0.02$) in the five patients whose left ventricular volume decreased by ≥20%. All five of these patients (four with anterior and one with inferior infarction) had residual perfusion of the infarct-related artery. There was no significant change in ejection fraction in patients with <20% change in left ventricular volume. However, in patients with anterior infarction who developed left ventricular dilation, ejection fraction decreased from 0.38 ± 0.10 to 0.30 ± 0.16 ($p = 0.05$). All but one of these patients had persistent total occlusion of the infarct artery without collateral filling. There was no significant change in ejection fraction in five patients with inferior infarction who developed left ventricular dilation.

Clinical course. Three patients had recurrent chest pain and a secondary rise in plasma creatine kinase consistent with infarct extension within 5 days of the initial infarction. All three had an increase in left ventricular volume by 1 month and all had a totally occluded infarct-related artery without collateral filling. None of the other 13 patients whose left ventricular volume increased by 20% or more had evidence of infarct extension.

During the month after infarction, 8 of the 16 patients with left ventricular dilation developed left ventricular failure (dyspnea of New York Heart Association class III or IV) and 6 were readmitted to the hospital (4 with pulmonary edema and 2 with ventricular tachycardia). In contrast, only 1 of the 24 patients without dilation developed left ventricular failure and none was readmitted to the hospital. There were no differences in treatment with nitrates, beta-adrenergic blockers or calcium antagonists between the patients who did and did not develop left ventricular dilation; however, patients with dilation were more frequently treated with digoxin or diuretics.

Figure 5. Combined relation of perfusion of the infarct-related artery and infarct size, measured by peak CK level, to change in left ventricular volume in the month after infarction. At each level of infarct size, patients with an unperfused infarct artery (**closed circles**) have a greater volume increase than do patients with a perfused infarct artery (**open circles**).



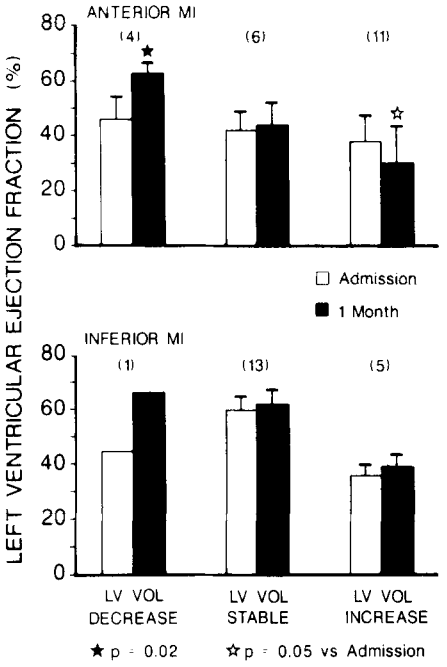


Figure 6. Relation between change in left ventricular volume (LV VOL) and change in left ventricular ejection fraction in the month after infarction (mean ± SD) in patients with anterior versus those with inferior myocardial infarction (MI).

Discussion

Left ventricular volume after myocardial infarction. This study examined the natural history of changes in left ventricular volume in the month after a first myocardial infarction in 40 patients who did not receive thrombolytic therapy or coronary angioplasty. Left ventricular dilation, defined as a ≥20% increase in volume, occurred in 16 patients (40%) and a decrease in volume of ≥20% occurred in 5 patients (12.5%). The degree of perfusion of the infarct-related artery was the most important predictor of change in ventricular volume, independent of infarct size. All patients with an unperfused infarct artery developed left ventricular dilation and at each level of infarct size patients with an unperfused infarct artery had a greater increase in volume than did patients with a perfused infarct artery. The effect of infarct artery perfusion was the same whether it was anterograde through either a subtotal lesion or collateral filling into a totally occluded artery.

Coronary angiography was performed 7 to 10 days after infarction. Other studies have demonstrated that 80 to 90% of patients studied within 4 hours of infarction have a totally occluded infarct-related artery (28). Thus, the majority of patients with a total occlusion of this artery at 10 days probably had persisting total occlusion from the time of infarction. Similarly, most patients with subtotal coronary occlusion at 10 days probably had at the time of infarction a totally occluded artery that spontaneously recanalized by the time of angiography.

The changes in global left ventricular volume documented in this study are consistent with the regional myocardial changes observed in previous animal (9–11) and human (5,6,29,30) studies. Infarct expansion, which can occur within 48 hours of infarction, appears to be the first event in the left ventricular remodeling process, which may continue after hospital discharge and involve both infarcted and noninfarcted myocardium (8) leading to global dilation of the left ventricular cavity.

The significant decrease in left ventricular volume observed in five patients, with an accompanying increase in ejection fraction, coincides with observations that early infarct expansion may completely or partially resolve in some patients (5,6). The volume decrease in our patients may represent resolution of such early infarct expansion. It is significant that all five of these patients had a well perfused infarct-related artery.

Role of infarct size. Infarct size has previously been considered to be the major determinant of left ventricular dilation (6,9,10). Direct measurements of infarct size in animal studies (31) have shown that impairment of ventricular function is related to extent of infarction. However there remains considerable variation in the relation between infarct size and subsequent infarct thinning and cavity dilation (9–11). Transmural infarction does appear to be a requirement for the development of dilation (9).

In humans, only indirect measures of infarct size are available. We have used two independent indexes of infarct size; peak creatine kinase and the QRS score derived from the 12 lead electrocardiogram. The peak creatine kinase level may be influenced by the state of perfusion of the infarct artery (32), but the QRS score, which correlates closely with pathologic measurements of infarct size (15,16), is likely to be independent of infarct artery perfusion. As in previous studies (5,6), we did not find a consistent relation between these indexes of infarct size and changes in ventricular volume. The relation between infarct size and change in volume might have been improved by more direct measurement of infarct size. However, the association between absent perfusion of the infarct artery and ventricular dilation is so strong that it is unlikely that the independent effect of infarct artery perfusion on volume would be invalidated by more direct measures of infarct size.

Role of infarct artery perfusion. Our findings suggest that perfusion of the infarct during the healing phase may be important in preventing continuing infarct expansion and subsequent left ventricular dilation. Conversely, lack of perfusion during the healing phase, particularly if the infarct is large, appears to predispose the patient to infarct expansion and left ventricular dilation. Other observations that fit this hypothesis include the association between persistent occlusion of the left anterior descending artery and aneurysm formation in patients with anterior infarction (33) and the observation in animal studies that, independent of infarct size, unperfused infarcts are more likely to expand than are perfused infarcts (34).

There are histologic differences between unperfused and reperfused infarcts that may underlie the differing propensity to dilation. Absence of perfusion is associated with coagulation necrosis whereas reperfusion causes contraction band necrosis (35). Recent evidence (36) suggests that the tensile strength of unperfused and reperfused infarct scars is different, although there is doubt as to whether this is significant at physiologic pressures. It is possible that reperfusion during the early healing phase may prevent the process of myocyte slippage and infarct thinning which results in infarct expansion and may lead to continuing ventricular dilation. Another possibility is that reperfusion may prevent expansion and dilation by preserving islands of epicardium.

Clinical implications. Previous studies (37,38) involving serial measurements of left ventricular function in postinfarction patients have reported a significant decrease in left ventricular ejection fraction in a small proportion of patients. Progressive left ventricular dilation, related to lack of perfusion of the infarct-related artery, appears to underlie this late deterioration in left ventricular function. The question then arises whether late deterioration in function may be prevented by maintaining perfusion of this artery.

Apart from the study by Nienaber et al. (39), which failed to demonstrate any effect of spontaneous recanalization on left ventricular function, most other studies have demonstrated a beneficial relation between patency of the infarct-related artery and left ventricular ejection fraction. De Feyter et al. (40) found that infarct artery patency was associated with a higher ejection fraction in patients with inferior infarction. Betriu et al. (41) found that total or near-total occlusion of the infarct artery was associated with a lower ejection fraction unless collateral vessels were present, and Blanke et al. (42) found that total occlusion of the infarct artery was associated with a decrease in ejection fraction. The differences between patients with a perfused and nonperfused infarct artery did not appear to merely result from differences in infarct size; however, none of these studies considered left ventricular volume when assessing left ventricular function. Our study demonstrates that late changes in ejection fraction reflect changes in left ventricular volume and thus result from remodeling of the left ventricle after infarction.

These results may have important implications for acute interventions in myocardial infarction. Early reperfusion is critical in achieving maximal salvage of jeopardized myocardium (43). Our data suggest that perfusion of the infarct-related artery may also be important during the healing phase to prevent subsequent left ventricular dilation. The possibility that reperfusion that is too late to prevent myocardial damage may still be beneficial in preventing late deterioration in left ventricular volume should be examined.

References

1. Kostuk WJ, Kazamias TM, Gander MP, et al. Left ventricular size after acute myocardial infarction: serial changes and their prognostic significance. Circulation 1973;47:1174–9.
2. Norris RM, Barnaby PF, Brandt PWT, et al. Prognosis after recovery from first acute myocardial infarction. Determinants of reinfarction and sudden death. Am J Cardiol 1984;53:408–13.
3. Henning H, Gilpin EA, Covell JW, et al. Prognosis after acute myocardial infarction: a multivariate analysis of mortality and survival. Circulation 1979;59:1124–33.
4. Nestico PF, Hakki AH, Iskandrian AS. Left ventricular dilatation. Prognostic value in severe left ventricular dysfunction secondary to coronary artery disease. Chest 1985;88:215–20.
5. Erlebacher JA, Weiss JL, Weisfeldt ML, Bulkley BH. Early dilation of the infarcted segment in acute transmural myocardial infarction: role of infarct expansion in acute left ventricular enlargement. J Am Coll Cardiol 1984;4:201–8.
6. Meizlish JL, Berger HJ, Plankey M, et al. Functional left ventricular aneurysm formation after acute anterior transmural myocardial infarction. N Engl J Med 1984;311:1001–6.
7. Warren SE, McKay RG, Royal HD, et al. Time course of left ventricular dilatation following acute myocardial infarction (abstr). Circulation 1985;72(suppl III):III-439.
8. Erlebacher JA, Weiss JL, Eaton LW, et al. Late effects of acute infarct dilation on heart size: a two dimensional echocardiographic study. Am J Cardiol 1982;49:1120–5.
9. Eaton LW, Bulkley BH. Expansion of acute myocardial infarction: its relationship to infarct morphology in a canine model. Circ Res 1981;49:80–8.
10. Hochman JS, Bulkley BH. Expansion of acute myocardial infarction: an experimental study. Circulation 1982;65:1446–50.
11. Roberts CS, Maclean D, Braunwald E, et al. Topographic changes in the left ventricle after experimentally induced myocardial infarction in the rat. Am J Cardiol 1983;51:871–6.
12. Pirolo JS, Hutchins GM, Moore GW. Infarct expansion: pathologic analysis of 204 patients with a single myocardial infarct. J Am Coll Cardiol 1986;7:349–54.
13. Nicklas JM, Becker LC, Bulkley BH. Repeated brief coronary occlusion: effects on regional shape, function and ultrastructure (abstr). Clin Res 1982;30:209A.
14. Palmeri ST, Harrison DG, Cobb FR, et al. A QRS scoring system for assessing left ventricular function after myocardial infarction. N Engl J Med 1982;306:4–9.
15. Ideker RE, Wagner GS, Ruth WK, et al. Evaluation of a QRS scoring system for estimating myocardial infarct size. II. Correlation with quantitative anatomic findings for anterior infarcts. Am J Cardiol 1982;49: 1604–14.
16. Roark SF, Ideker RE, Wagner GS, et al. Evaluation of a QRS scoring system for estimating myocardial infarct size. III. Correlation with quantitative anatomic findings for inferior infarcts. Am J Cardiol 1983;51: 382–9.
17. Pavel DG, Zimmer AM, Patterson VN. In vivo labelling of red blood cells with 99m Tc: a new approach to blood pool visualisation. J Nucl Med 1977;18:305–8.
18. Hutton BF, Cormack J, Fulton RR. A software package for the analysis of gated cardiac blood pool studies. Australas Phys Eng Sci Med 1982;5:182–4.
19. Slutsky R, Karliner J, Ricci D, et al. Left ventricular volumes by gated equilibrium radionuclide angiography: a new method. Circulation 1979;60:556–64.
20. Massie BM, Kramer BL, Gertz EW, Henderson SG. Radionuclide measurement of left ventricular volume: comparison of geometric and counts-based methods. Circulation 1982;65:725–30.
21. Dehmer GJ, Lewis SE, Hillis LD, et al. Non-geometric determination of left ventricular volumes from equilibrium blood pool scans. Am J Cardiol 1980;45:293–300.
22. Jeremy RW, Tokuyasu Y, Choong CYP, et al. The reproducibility of non-geometric analysis of cardiac output and left ventricular volume by radionuclide angiography. Am Heart J 1985;110:1020–6.
23. TIMI Study Group. The thrombolysis in myocardial infarction (TIMI) trial. Phase 1 findings. N Engl J Med 1985;312:932–6.
24. Hecht HS, Aroesty JM, Morkin E, et al. Role of the coronary collateral circulation in the preservation of left ventricular function. Radiology 1975;114:305–13.
25. Roubin GS, Shen WF, Kelly DT, Harris PJ. The QRS scoring system for estimating myocardial infarct size: clinical, angiographic and prognostic correlations. J Am Coll Cardiol 1983;2:38–44.
26. Snedecor GW, Cochran WG. Statistical Methods. 7th ed. Iowa City: Iowa State University Press, 1982:141–3.
27. Dixon WJ, Brown MB, Engelman L, et al., eds. BMDP Statistical Software. Los Angeles: University of California Press, 1983.
28. DeWood MA, Spores J, Notske R, et al. Prevalence of total coronary occlusion during the early hours of transmural myocardial infarction. N Engl J Med 1980;303:898–902.
29. Hutchins GM, Bulkley BH. Infarct expansion versus extension: two different complications of acute myocardial infarction. Am J Cardiol 1978;41:1127–32.
30. Eaton LW, Weiss JL, Bulkley BH, et al. Regional cardiac dilatation after acute myocardial infarction. Recognition by two-dimensional echocardiography. N Engl J Med 1979;300:57–62.
31. Fletcher PJ, Pfeffer JM, Pfeffer MA, Braunwald E. Left ventricular diastolic pressure-volume relations in rats with healed myocardial infarction. Effects on systolic function. Circ Res 1981;49:618–26.
32. Blanke H, von Hardenberg D, Cohen M, et al. Patterns of creatine kinase release during acute myocardial infarction after nonsurgical reperfusion: comparison with conventional treatment and correlation with infarct size. J Am Coll Cardiol 1984;3:675–80.
33. Forman MB, Kopelman HA, Collins HW, et al. Poor collaterals and single vessel disease predispose to aneurysm formation after anterior myocardial infarction (abstr). Circulation 1985;72(suppl III):III-439.
34. Hochman JS, Choo H. Coronary reperfusion inhibits infarct expansion independent of myocardial salvage (abstr). Circulation 1985;72(suppl III):III-67.
35. Hutchins GM, Bulkley BH. Correlation of myocardial contraction band necrosis and vascular patency; a study of coronary artery bypass graft anastamoses at branch points. Lab Invest 1977;36:642–8.
36. Connelly CM, Vogel WM, Wiegner AW, et al. Effects of reperfusion after coronary artery occlusion on post-infarction scar tissue. Circ Res 1985;57:562–77.
37. Kostuk WJ, Ehsani AA, Karliner JS, et al. Left ventricular performance after myocardial infarction assessed by radioisotope angiocardiography. Circulation 1973;47:242–9.
38. Reduto LA, Berger HJ, Cohen LS, et al. Sequential radionuclide assessment of left and right ventricular performance after acute transmural myocardial infarction. Ann Intern Med 1978;89:441–7.
39. Nienaber CA, Spielmann RP, Rozza A, et al. Prevalence of spontaneous coronary thrombolysis and its relation to left ventricular function. Eur Heart J 1985;6(suppl E):145–53.
40. de Feyter PJ, van Eenige MJ, van der Wall EE, et al. Effects of spontaneous and streptokinase induced recanalization on left ventricular function after myocardial infarction. Circulation 1983;67:1039–44.
41. Betriu A, Castaner A, Sanz GA, et al. Angiographic findings 1 month after myocardial infarction: a prospective study of 259 survivors. Circulation 1982;65:1099–1104.
42. Blanke H, Cohen M, Karsch KR, et al. Prevalence and significance of residual flow to the infarct zone during the acute phase of myocardial infarction. J Am Coll Cardiol 1985;5:827–31.
43. Mathey DG, Sheehan FH, Schofer J, Dodge HT. Time from onset of symptoms to thrombolytic therapy: a major determinant of myocardial salvage in patients with acute transmural infarction. J Am Coll Cardiol 1985;6:518–25.